PB88-162383

Health and Environmental Effects Profile for MCPP

(U.S.) Environmental Protection Agency
Cincinnati, OH

Jan 84

U.S. Department of Commerce
National Technical Information Service
NTIS

PB88-162383

EPA/600/X-84/241
January 1984

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR MCPP

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
CINCINNATI, OH 45268

REPRODUCED BY
U.S. DEPARTMENT OF COMMERCE
NATIONAL TECHNICAL
INFORMATION SERVICE
SPRINGFIELD, VA. 22161

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA/600/X-84/241	2.
3. RECIPIENT'S ACCESSION NO. PB88 162383/AS	
4. TITLE AND SUBTITLE Health and Environmental Effects Profile for MCPP	5. REPORT DATE January 1984
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS Same as box 12.	10. PROGRAM ELEMENT NO.
	11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268	13. TYPE OF REPORT AND PERIOD COVERED
	14. SPONSORING AGENCY CODE EPA/600/22

15. SUPPLEMENTARY NOTES

16. ABSTRACT

The Health and Environmental Effects Profile for MCPP was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. MCPP has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for MCPP is 3.4×10^{-3} (mg/kg bw/day) for oral exposure.

10034

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 42
	20. SECURITY CLASS (This page) Unclassified	22. PRICE

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Bail was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
Carcinogen Assessment Group
Exposure Assessment Group
Reproductive Effects Assessment Group

Editorial review for the document series was provided by:
Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office, Cincinnati, OH

Technical support services for this document series were provided by:
Bette Zwayer, Pat Daunt, Karen Mann, and Jacky Bohanon
Environmental Criteria and Assessment Office, Cincinnati, OH.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CHEMICAL ABSTRACTS SERVICE REGISTRY NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION DATA	3
1.4.	USE DATA	3
2.	ENVIRONMENTAL FATE AND TRANSPORT PROCESSES	5
2.1.	AIR	5
2.2.	WATER	6
2.3.	SOILS	8
3.	EXPOSURE	11
4.	PHARMACOKINETICS	12
4.1.	ABSORPTION	12
4.2.	DISTRIBUTION	12
4.3.	METABOLISM	12
4.4.	EXCRETION	13
5.	EFFECTS	14
5.1.	CARCINOGENICITY	14
5.2.	MUTAGENICITY	14
5.3.	TERATOGENICITY	14
5.4.	OTHER REPRODUCTIVE EFFECTS	14
5.5.	CHRONIC AND SUBCHRONIC TOXICITY	16
5.6.	OTHER RELEVANT INFORMATION	17
6.	AQUATIC TOXICITY	19
6.1.	ACUTE	19
6.2.	CHRONIC	19
6.3.	PLANT EFFECTS	20
6.4.	RESIDUE	20
6.5.	OTHER RELEVANT INFORMATION	20
7.	EXISTING GUIDELINES AND STANDARDS	21
7.1.	HUMAN	21
7.2.	AQUATIC	21
8.	RISK ASSESSMENT	22
9.	REFERENCES	24
APPENDIX:	Literature Searched	33

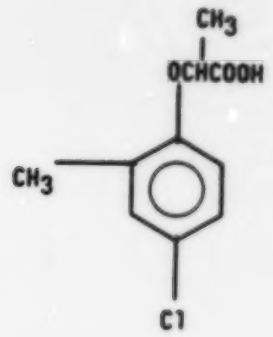
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
bw	Body weight
DNA	Deoxyribonucleic acid
GI	Gastrointestinal
i.p.	Intraperitoneal
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
LOAEL	Lowest-observed-adverse-effect level
NOAEL	No-observed-adverse-effect level
NOEL	No-observed-effect level
ppb	Parts per billion
ppm	Parts per million
TLC	Thin-layer chromatography

1. INTRODUCTION

1.1. STRUCTURE AND CHEMICAL ABSTRACTS SERVICE REGISTRY NUMBER

2(2-Methyl-4-chlorophenoxy)propionic acid (MCPP) is also known as mecoprop, 4-chloro-2-methylphenoxy-α-propionic acid and 2-(4-chloro-2-methylphenoxy) propionic acid. The structure of MCPP is shown below:



The molecular formula for MCPP is $C_{10}H_{11}ClO_3$ and the molecular weight is 214.66. The Chemical Abstracts Service Registry Number is 93-65-2.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

MCPP is a white crystalline solid when pure; it is available commercially as free acid, as salts and as esters. The melting point of MCPP is 94-95 and the water solubility (mg/ℓ) is 620 at 20°C. MCPP will dissociate in water 99% at pH values >5. Stevens and Grorud (1979) identified a number of impurities in the acetic acid homologue, MCPA. Given the similarities in manufacturing, it is likely that some of the same impurities will be present in MCPP. Table 1-1 lists the impurities found in MCPA. Since MCPA is the acetic acid derivative of the phenoxy moiety, while MCPP is the propionic acid derivative, impurities in MCPA as an acetic acid may be present in MCPP as a propionic acid.

TABLE 1-1

Analysis of Commercial MCPA*

Compound	Nominal Percent in MCPA
Phenolic impurities	
2-Methyl phenol	0-0.1
3-Methyl phenol	0-0.1
2-Chlorophenol	0-0.1
4-Chlorophenol	0-0.1
2-Chloro-6-methyl phenol	0-0.1
4-Chloro-2-methyl phenol	0-0.5
4-Chloro-3-methyl phenol	0-1
4-chloro-2,6-dimethyl phenol	0-0.5
2,4-Dichloro-6-methyl phenol	0-0.5
MCPA	90-99
Phenoxy acid impurities	
2-Methylphenoxyacetic acid	0-2.5
3-Methylphenoxyacetic acid	0-0.5
2-Chlorophenoxyacetic acid	0-0.5
4-Chlorophenoxyacetic acid	0-0.5
2-Chloro-6-methyl phenoxyacetic acid	0-4
4-Chloro-2,6-dimethylphenoxyacetic acid	0-0.5
2,4-Dichloro-6-methylphenoxyacetic acid	0-3
MCPA	90-100

*Source: Stevens and Grorud, 1979

1.3. PRODUCTION DATA

MCPP is produced by reacting 2-chloropropionic acid with sodium 4-chloro-o-cresolate. This gives two enantiomers of which only the (+)-form is active. No recent production volume data were found in the available literature. In 1975, 0.1 million pounds were produced (SRI, 1976). Manufacturing facilities are listed in Table 1-2.

1.4. USE DATA

MCPP is a hormone type herbicide that is effective against broadleaf plants in cereals (BCPC, 1977). MCPP is used as the acid, salts and esters.

TABLE 1-2

MCPP Manufacturing Facilities

Manufacturer	Location
W.A. Cleary Corp.	Somerset, NJ
Diamond Shamrock Corp.	Tuscaloosa, AL
Rhone-Poulenc, Inc.	Portland, OR
Riverdale Chemical Co.	Chicago Heights, IL

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. AIR

No information was found in the available literature concerning the atmospheric chemistry of MCPP. Hendry and Kenley (1979) presented the rate constants and estimated half-lives for reactions with hydroxyl radicals of several alkyl- and fluoro-substituted benzenes, anisole and o-cresol, which have similarities in structure. Based on these values, MCPP would be expected to have an atmospheric half-life of <1-10 days. A similar estimation for reaction with ozone is more difficult, since fewer data for this reaction were presented in Hendry and Kenley (1979); however, based on their data, the half-life for MCPP would probably be <10 days.

No information concerning the vapor phase photolysis of MCPP was found in the available literature. Que Hee and Sutherland (1979) photolyzed 2,4-dichlorophenoxyacetic acid butyl ester (2,4-D butyl ester) under simulated environmental conditions. The ester was in both the pure liquid and vapor phase. The authors reported an approximate half-life of 300 hours; given the similarities in structure, a half-life of the same order would be expected for MCPP.

The vapor phase transport of MCPP in air will not be an important process for the free acids or their salts because of their low vapor pressure. The esters of these acids, however, are volatile to varying degrees and can be transported long distances in the vapor phase. In addition to vapor phase transport, spray droplet transport will be a factor, since MCPP is sometimes applied by spray. However, spray drift generally is limited to a few kilometers (Bogyo et al., 1981).

No information was found in the available literature concerning the vapor drift of MCPP esters. Considerable information, however, is available

for 2,4-D esters; these esters have densities and other physical properties similar to MCPP esters and would be expected to behave in an analogous fashion. A number of factors influence the vapor drift of chlorophenoxy herbicides, including the surface area of the herbicide exposed to air, the absorptive and adsorptive characteristics of the surface to which the ester is adhering, the vapor pressure of the ester, the carrier solvent and the additives present in the mix (Bogyo et al., 1981). In general, the esters with lower molecular weights are more volatile (i.e., they have higher vapor pressure). Vapor pressures for the esters of 2,4-D vary from 10^{-3} torr for the methyl ester to $<10^{-6}$ torr for the esters with higher molecular weights (Jensen and Schall, 1966; Hamaker and Kerlinger, 1969; Flint et al., 1968; Kybett et al., 1976). The distances traveled by ester vapors appear to be on the order of "tens of miles" (Reisinger and Robinson, 1976, 1977) for the highly volatile esters. This transport is dependent on weather conditions and is aided by weak frontal systems. Grover et al. (1976) reported that the highest atmospheric concentrations of chlorophenoxy herbicide esters came in the spraying season, and that low concentrations were present during or immediately after rain (this indicates that these esters are washed out of the air).

2.2. WATER

The only important aqueous chemical process for MCPP esters is hydrolysis. No information concerning the rates for this process was found in the available literature; however, Mill (1980) reported on the hydrolysis of 2,4-D butyl ester. At pH 4, the half-life is on the order of 2 years, while at pH 7, the half-life is ~8 days. A similar type of pH-hydrolysis half-life relationship is expected for MCPP esters, since they have similar structures. Zepp et al. (1975) used theoretical considerations and

structure-activity relationships to estimate the hydrolysis half-lives of 2,4-D esters. They reported the half-lives for the methyl, isopropyl, n-butyl, n-octyl, isooctyl and butoxyethyl esters to be 44, 710, 220, 220, 1500 and 26 days, respectively, at pH 6; at pH 9, the half-lives were 1.1, 17.0, 5.2, 5.2, 37 and 0.6 hours, respectively. Rodgers and Stalling (1972) reported that the addition of fish to an aquarium decreased the hydrolysis half-life of 2,4-D butoxyethyl ester from ~40 days to <4 days. Again, a similar behavior is expected for MCPP in that the esters with high molecular weights will hydrolyze more slowly than those with low molecular weights, and that fish will decrease the hydrolysis half-lives of esters in water. Nevertheless, exact half-lives for MCPP esters cannot be estimated from these data; since the esters have similar structures, however, the half-lives are probably on the same order of magnitude.

Soderquist and Crosby (1975) reported that when MCPA (a phenoxy herbicide of similar structure) was photolyzed in water using fluorescent light (>290 nm, pH 9.8), 4-chloro-2-methylphenol, 4-chloro-2-formylphenol and o-cresol were formed. Strekozov et al. (1979) also reported formation of 4-chloro-2-methylphenol when MCPA was photolyzed. At pH 8.3, the half-life of MCPA in sunlight is 20-24 days (Soderquist and Crosby, 1975).

Virtanen et al. (1979) studied MCPA dissipation in a model ecosystem containing water, soil, sand, plants (three genera), fish, mice and snails. MCPA sodium salt was sprayed on the soil and watered 3 times during the study. MCPA concentrations declined in the soil (0.190 to 0.114 ppm) over 12 days, but increased in the water (0.045-0.156 ppm). After 22 days, 20% of the applied MCPA remained. Soderquist and Crosby (1975) reported that MCPA in rice paddy water in the dark was 99% removed in 14 days by aquatic organisms after application of 0.98 kg/ha MCPA dimethylamine salt.

2.3. SOILS

Very little information concerning MCPP in soil was found in the available literature. Smith and Hayden (1981) treated Saskatchewan soils with MCPA, MCPP and MCPB (the butyric acid homologue of MCPP) to determine their persistence. In three different soils, MCPA, MCPB and MCPP had half-lives of 5-7, 6-7 and 7-9 days, respectively. Kirkland and Fryer (1972) found MCPP somewhat more persistent than MCPA or MCPB. Detectable residues lasted 32 days in soil previously untreated with chlorophenoxy herbicides, but only 16 days after the soils had been treated. MCPA and MCPB persisted between 4 and 16 days for soils pretreated and not treated with chlorophenoxy herbicides, respectively. Based on these data, MCPP appears to have soil dissipation rates similar to MCPA and MCPB. Since much more information is present for MCPA, a summary of the data available for it is presented; the dissipation of MCPP and the factors affecting it are probably very similar.

MCPA appears to be degraded rapidly in most soils, with half-lives varying from a few days to ~1 month in both laboratory and field studies (Kirkland and Fryer, 1972; Soulas et al., 1983; Stark, 1983; Smith and Hayden, 1980; Sattar, 1982; Sattar and Paasivirta, 1980; Smith and Hayden, 1981; Smith, 1982). Dissipation to below the detection limits requires a longer period, varying from 4 days (Kirkland and Fryer, 1972) to over 32 weeks (Sattar, 1982). Variations depend on a number of factors, including application rates (higher concentrations give lower removal rates), whether or not the soil has been previously treated with chlorophenoxy herbicides (previous treatment increases dissipation rates), organic matter content (higher organic matter content lowers removal rates), soil moisture (low moisture content slows degradation), temperature (higher temperatures increase degradation rates) and soil pH (degradation is most rapid between

pH 5.5 and 8.5). For the most part, however, it appears that the magnitude of these variations are such that degradation rates will be independent of soil type and bacterial population (Loos et al., 1979).

MCPA is expected to be mobile in most soils. Helling (1971) and Helling and Turner (1968), using soil TLC, reported that MCPA was mobile (R_f 0.6-1.0) on Chillium silt loam (3.1% organic matter), Lakeland sandy loam (0.9% organic matter) and silty clay loam (1.4% organic matter)soils. Herzel and Schmidt (1979) reported that phytotoxic levels of MCPA leached 30 cm in a sandy soil column diluted with 50 cm of water. Despite its leachability, MCPA has only rarely been detected in groundwater. In 237 wells in Ontario, Canada, sampled between 1969 and 1978 (393 samples), MCPP was present in only nine (Frank et al., 1979). Two were contaminated between 0.01 and 0.1 ppb, 2 between 0.1 and 1.0 ppb, 4 between 11 and 100 ppb and 1 >10,000 ppb. The herbicide entered the wells either by direct spill of the concentrate or diluted mixture, or by spray drift. An accidental spill of a 40% solution of MCPA (1000 ℓ) in Czechoslovakia resulted in MCPA contamination of wells 150 m from the spill site. Concentrations from 0.1-5.4 ppm were detected, and persisted from 8-11 months after the spill.

The lack of information concerning detectable residues of MCPA, MCPP or MCPB in groundwater may be because of its rapid biodegradation (see above); however, there is insufficient information concerning use and use patterns to permit a more positive conclusion about the leaching potential of MCPP.

A number of the metabolites of MCPA have been determined both from pure microbial cultures and from field studies. It is likely that similar or identical metabolites will result from MCPP metabolism. Gaunt and Evans (1971a,b) and Gamar and Gaunt (1971) found that a soil Pseudomonad (N.C.I.B. 9340, National Collection of Industrial Bacteria) and cell-free extracts

prepared from this bacterium metabolized MCPA, yielding, initially, 5-chloro-o-cresol and 5-chloro-3-methyl catechol. Further work suggested the following metabolic pathway: MCPA → 5-chloro-o-cresol → 5-chloro-3-methylcatechol → cis-cis-γ-chloro-α-methylmuconate → γ-carboxymethylene-α-methyl-$\Delta^{\alpha,\beta}$-butenolide → γ-hydroxy-α-methylmuconate. 4-Chloro-o-cresol was identified as a product of a soil bacterium Flavobacterioni peregrinum by Bollag et al. (1967) and a soil Pseudomonas sp. by Kilpi et al. (1980, 1983). Sattar (1981) also identified this compound as well as 5-chloro-3-methylcatechol in whole soils treated with MCPA. Bollag et al. (1967) also reported that an Arthrobacter sp. isolated from soil could dehalogenate MCPA and several catechols. Kilpi et al. (1980) reported isolating 2-hydroxy-muconic acid when a Pseudomonas sp. HV3 was incubated with MCPA. Alcaligenes paradoxus has also been reported to metabolize MCPA (Fisher et al., 1978; Jensen and Petersen, 1952a,b; Steenson and Walker, 1956).

Several researchers (Faulkner and Woodcock, 1965; Nakajima et al., 1973; Torstensson et al., 1975) have reported that fungi Aspergillus niger, Fusarium culmorum, Gloeosporum kaki, G. olivarum, Mucor sp., Penicillium sp., Schizophyllum commune, Verticillium spp. and Zygorhynchus moelleri metabolize MCPA; 4-chloro-5-hydroxy-2-methylphenoxyacetate and 2-(2-methyl-4-chlorophenoxy)ethanol are two metabolites.

No information was found in the available literature concerning the metabolic transformation pathways of MCPB; however, since MCPA and MCPB are structurally similar, it is anticipated that MCPB will degrade similarly.

3. EXPOSURE

No information was found in the available literature, including STORET, concerning environmental residues other than those presented in Chapter 2. From these it appears that widespread exposure to MCPP does not occur, and that when exposure occurs, it is the result of an accidental spill rather than persistence.

4. PHARMACOKINETICS

4.1. ABSORPTION

That MCPP is absorbed from the GI tract can be inferred from the case study of a 39-year-old man who ingested ~70 mL (estimated from plasma concentrations) of a weedkiller containing 20% MCPP and 10% 2,4-dichlorophenoxyacetic acid (2,4-D) (Prescott et al., 1979). Two hours after the weedkiller was ingested, the plasma concentration of MCPP was 751 µg/mL, and decreased slowly with a half-life of ~40 hours. Alkaline diuresis moderately increased the elimination of plasma concentration of MCPP. The half-lives of plasma clearance just before, during (~96 hours after ingestion) and after the subject's urine became alkaline as a result of therapeutic alkaline diuresis were 24, 11 and 28 hours, respectively. Four days after the weed-killer was ingested, the plasma concentration of MCPP was 100 µg/mL.

Gurd et al. (1965) reported that MCPP is rapidly absorbed from the GI tract of rats and mice, but poorly absorbed through the skin of mice, even following damage to the skin.

4.2. DISTRIBUTION

MCPP was found at a plasma concentration of 751 µg/mL in a 39-year-old man 2 hours after he had ingested 70 mL of a weedkiller containing 20% MCPP and 10% 2,4-D (see Section 4.1.) (Prescott et al., 1979). No other tissue levels of MCPP were reported.

4.3. METABOLISM

Prescott et al. (1979) reported that MCPP underwent extensive metabolism in a 39-year-old man who had ingested ~70 mL of a weedkiller containing 20% MCPP and 10% 2,4-D (see Section 4.1.). Of the total amount of MCPP recovered in the urine (7.64 g, total time not specified), 65% was reported to be acid-labile conjugates.

4.4. EXCRETION

Prescott et al. (1979) reported urinary excretion data for MCPP from a 39-year-old man who had ingested ~70 mL of a weedkiller containing 20% MCPP and 10% 2,4-D (see Section 4.1.). Urinary excretion of MCPP during the first 72 hours after ingeston was 1.42 g, and from 72-134 hours after ingestion it was 2.15 g. Total urinary excretion of MCPP (total time not specified) was 7.64 g.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of MCPP could not be located in the available literature; this compound is not scheduled for carcinogenicity testing by the National Toxicology Program (NTP, 1983).

5.2. MUTAGENICITY

MCPP has been tested for mutagenicity in bacterial and yeast systems. These mutagenicity tests are summarized in Table 5-1.

MCPP was negative in reverse mutation assays with Salmonella typhimurium (Andersen et al., 1972; Carere et al., 1976; Moriya et al., 1983) and with Escherichia coli (Moriya et al., 1983), and in the forward mutation assay with Streptomyces coelicolor (Carere et al., 1976). MCPP elicited a positive response in the mitotic gene conversion assay with Saccharomyces cerevisiae, but induction of mitotic gene conversion at two separate loci (ade-2 and his-I) by MCPP occurred only at high doses, which resulted in low cell survival (Parry, 1973). MCPP was negative in forward mutation, nondisjunction and mitotic crossing-over assays with Aspergillus nidulans (Aulicino et al., 1976).

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of MCPP could not be located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Seiler (1979) examined the effect of MCPP on testicular DNA synthesis in male mice. The animals were given an i.p. dose of [^{14}C]-methyl-labeled thymidine, followed an hour later by 200 mg MCPP/kg bw orally; [^{3}H]methyl-labeled thymidine was administered intraperitoneally 3 hours after

TABLE 5-1

Mutagenicity Testing of MCPP

Assay	Indicator Organism	Application	Concentration or Dose	Activation System	Response	Comment	Reference
Reverse mutation	S. typhimurium TA98 TA100 TA1535 TA1537 TA1538	plate incorporation	<5000 µg/plate	± S-9	- - - - -	NC	Moriya et al., 1983
Reverse mutation	S. typhimurium 6 histidine-requiring strains, NOS	spot test	~1-5 µl	none	-	NC	Andersen et al., 1972
Reverse mutation	S. typhimurium TA1535 TA1536 TA1537 TA1538	plate incorporation	NR	NR	-	NC	Carere et al., 1976
Reverse mutation	Escherichia coli, WP2 hcr	plate incorporation	<5000 µg/plate	± S-9	-	NC	Moriya et al., 1983
Forward mutation	Streptomyces coelicolor	plate incorporation	NR	NR	-	NC	Carere et al., 1976
Mitotic gene conversion	Saccharomyces cerevisiae, diploid strain	suspension cultures	742-6678 ppm	none	+	MCPP induced mitotic gene conversion at two separate loci (ade-2 and his-1) only at low cell survival levels.	Parry, 1973
Forward mutation	Aspergillus nidulans	spot test	NR	NR	-	NC	Aulicino et al., 1976
Nondisjunction	A. nidulans, diploid strain, NOS	spot test or plating conidia	NR	NR	-	NC	Aulicino et al., 1976
Mitotic crossing-over	A. nidulans, diploid strain, NOS	spot test or plating conidia	NR	NR	-	NC	Aulicino et al., 1976

NR = Not reported

NC = No comment

NOS = Not otherwise specified

administration of the test compound, and the animals were killed 0.5 hour later. Treatment with MCPP resulted in significant ($p<0.01$) inhibition of testicular DNA synthesis (60% inhibition).

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Verschueren et al. (1975) administered MCPP (92-93% pure) ad libitum in the diet to weanling SPF-Wistar rats (10 of each sex/dose level) at concentrations of 0, 50, 400 or 3200 ppm for 90 days. Neither increased mortality nor histopathologic abnormalities related to treatment were observed. At the highest dose level, decreased body weight gain was accompanied by decreased food consumption. Treatment-related effects on blood composition at the highest dose level included significantly ($p<0.05$) decreased hemoglobin content, erythrocyte count, hematocrit (males only) and leukocyte count (males only). At the 400 ppm level, males had significantly ($p<0.05$) reduced erythrocyte counts; at the 50 ppm level, males had significantly ($p<0.05$) decreased hematocrits. At the highest dose level, blood alkaline phosphatase activity was significantly ($p<0.01$) decreased for both sexes, and females had significantly ($p<0.05$) decreased levels of neutrophil cells. Several relative organ weights were found to be significantly ($p<0.05$) increased at the 3200 ppm dose level. Verschuuren et al. (1975) indicated that these increases may have been related directly to decreased body weights. Relative increased organ weights at the highest dose level included the liver, kidneys, testes and brain of males, and the liver, kidneys, heart, thyroid and brain of females. Male kidney and female liver weights were significantly ($p<0.01$) increased at the 400 ppm level.

Gurd et al. (1965) administered the diethanolamine salt of MCPP at concentrations of 0, 100, 400, 1000 or 2500 ppm in the diet to rats (5 of each sex/dose level) for 7 months. Reduced body weight gain was reported for

males at the 1000 ppm level and for both sexes at the 2500 ppm level. Reduced food consumption was reported for males on the 2500 ppm diet only during the first month of administration. For both sexes of rats on diets containing ≥400 ppm MCPP diethanolamine salt, reduced erythrocyte counts, hemoglobin and packed cell volume were observed. Increased liver-to-body-weight ratios occurred in females ingesting ≥400 ppm MCPP diethanolamine salt, and in males at the highest dose level. Both sexes at all treatment levels had increased kidney-to-body-weight ratios. Although not quantified, the increase in kidney weight was described as slight for animals in the 100 ppm group. The only histological change occurred at the 2500 ppm level. Almost every rat at this dose level had enlarged hepatic parenchymal cells with homogeneous eosinophilic cytoplasm.

5.6. OTHER RELEVANT INFORMATION

The oral LD_{50}s for MCPP to rats and mice are 1210 and 650 mg/kg, respectively (Verschuuren et al., 1975; NIOSH, 1983). The i.p. LD_{50} for MCPP to rats is 402 mg/kg (Verschuuren et al., 1975), and the dermal LD_{50} to rabbits is 900 mg/kg (NIOSH, 1983).

Verschuuren et al. (1975) applied MCPP (29-93% pure) dermally as an aqueous paste to the shaved backs and flanks of chinchilla rabbits at levels of 0, 0.5, 1.0, or 2.0 g/kg bw, 5 times/week, for 3 weeks. Decreased body weight gain and dose-related erythema were seen at all treatment levels. There was no effect on hematologic indices, relative organ weights, or histologic parameters.

Prescott et al. (1979) reported the case study of a 39-year-old man who had ingested ~70 ml of a weedkiller containing 20% MCPP and 10% 2,4-D. Effects relating to ingestion of the weedkiller included prolonged deep

coma, pyrexia, hyperventilation, hypoxemia, myotonia, skeletal muscle damage and electrocardiographic changes consistent with cardiomyopathy. The patient recovered 5 days after ingestion, following alkaline diuretic therapy.

6. AQUATIC TOXICITY

6.1. ACUTE

The acute toxicity of MCPP formulations has been determined with four fish species and two invertebrate species. Schneider (1979) reported that 180 ppm of a formulation containing 27% MCPP acid at 27% 2,4-D acid was not lethal in 96 hours to bluegills (Lepomis macrochirus) or in 24 hours to waterfleas (Daphnia magna). Similarly, another formulation containing 17.3% MCPP acid and 17.3% 2,4-D acid was not lethal in 96 hours in either bluegills or rainbow trout (Salmo gairdneri) at 180 ppm.

Linden et al. (1979) measured the acute toxicity of a MCPP potassium salt formulation (640 g active ingredient/ℓ) to two brackish water species. The 96-hour LC_{50} was 115 mg/ℓ for bleak (Alburnus alburnus) and 97 mg/ℓ for copepods (Nitocra spinipes). Alabaster (1969) measured the 48-hour LC_{50} of two MCPP formulations to harlequin fish (Rasbora heteromorpha). The 48-hour LC_{50} was 248 ppm for Tordon C (30% MCPP and 0.94% picloram) and 11.0 ppm for NPH 1313 (40% MCPP and 10% ioxynil). The LC_{50} value for NPH 1313 is much lower than the values for any of the other MCPP formulations. The toxicity of NPH 1313 is probably caused by ioxynil, because Alabaster (1969) found that the 48-hour LC_{50} of ioxynil alone to harlequin fish was 0.35 ppm. With the exception of NPH 1313, the available data indicate that MCPP formulations have a low acute toxicity to fish and invertebrates. Data concerning the acute toxicity of technical grade MCPP could not be located in the literature searched.

6.2. CHRONIC

Pertinent data concerning the chronic effects of MCPP on aquatic organisms could not be located in the literature searched.

6.3. PLANT EFFECTS

The only available information concerning the effects of MCPP on aquatic plants was provided by Cornet (1968), who reported that a mixture of MCPP, MCPA, 2,4-D and picloram herbicides (in a ratio of 115:15:15:1, respectively) provided temporary control of pondweed (Potamogeton natans) at treatment levels of 1.9 kg/ha in rice fields.

6.4. RESIDUE

Pertinent data concerning residue of MCPP in aquatic organisms could not be located in the literature searched.

6.5. OTHER RELEVANT INFORMATION

Kenaga (1980) utilized published water solubility data and regression equations to estimate the fish BCF for MCPP acid and the potassium and diethamolamine salts of MCPP. The predicted BCF values were 16, 0.3 and 0.4, respectively, based on water solubilities of 620 ppm, 795,000 ppm and 580,000 ppm.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Pertinent data regarding the existing guidelines and standards for human exposure to MCPP could not be located in the available literature.

7.2. AQUATIC

Existing guidelines and standards for MCPP in the aquatic environment could not be located in the available literature.

8. RISK ASSESSMENT

The lack of a cancer bioassay for MCPP precludes the derivation of a cancer-based quantitative risk value. MCPP was negative in the reverse mutation assay with Salmonella typhimurium (Andersen et al., 1972; Carere et al., 1976; Moriya et al., 1983), and in the forward mutation assay with Streptomyces coelicolor (Carere et al., 1976). MCPP elicited a positive response in the mitotic gene conversion assay with Saccharomyces cerevisiae (Parry, 1973). MCPP was negative in forward mutation, nondisjunction and mitotic crossing-over assays with Aspergillus nidulans (Aulicino et al., 1976). Oral administration of 200 mg MCPP/kg bw to mice significantly inhibited testicular DNA synthesis (Seiler, 1979).

Verschuuren et al. (1975) and Gurd et al. (1965) noticed similar effects on blood parameters and relative kidney and liver weights in rats given diets containing MCPP and MCPP diethanolamine, respectively. The Verschuuren et al. (1975) study yields a NOAEL for reduced hematocrit in males exposed for 90 days to MCPP at 50 ppm. The 50 ppm exposure was considered a NOAEL, because at the next exposure level, 400 ppm, the hematocrit value was not significantly decreased, while only at the highest exposure level, 3200 ppm, were hematocrit and other hematologic parameters significantly altered. The Gurd et al. (1965) study reports a NOAEL for slight increased kidney-to-body-weight ratios in both sexes exposed for 7 months to 100 ppm MCPP diethanolamine (equivalent to 67 ppm MCPP). Using the longer term study of Gurd et al. (1965), which provides the highest NOAEL, an ADI can be derived. In view of the absence of food consumption data, the assumption that a rat consumes a daily amount of food equivalent to 5% of its body weight must be made. Using this assumption, an equivalent dose of 3.4 mg/kg bw/day can be calculated. An ADI value of 0.235 mg/day for a 70 kg human can be calcu-

lated by multiplying the animal dose by an assumed human body weight of 70 kg and applying an uncertainty factor of 1000 to convert from experimental animal data to human data, to protect the more sensitive individuals of a population and to account for the less than chronic exposure used in the study of Gurd et al. (1965). The diethanolamine salt of MCPP was used in the derivation of this ADI; the absorption of the diethanolamine salt may not be identical to that of MCPP. There is, however, little difference in the ADI if the study of Verschuuren et al. (1975) is used to derive the ADI. Using the same assumptions as previously described and an uncertainty factor of 1000, an ADI of 0.175 mg/day can be calculated. Since the ADI values derived from both studies are similar and support each other, the ADI derived from the highest NOAEL, 0.235 mg/day, is the most appropriate.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter.

The CBI Appendix to this document reviews other animal data, which support the ADI derived above. A NOAEL of 4 mg/kg bw/day was observed in a study in which dogs were fed MCPP in the diet for 13 weeks. Slight reductions in erythrocyte count, hematocrit and blood hemoglobin level were observed at 16 mg/kg bw/day in the same study.

9. REFERENCES

Alabaster, J.S. 1969. Survival of fish in 164 herbicides, insecticides, fungicides, wetting agents, and miscellaneous substances. Int. Pest. Control. 11(2): 29-35.

Andersen, K.J., E.G. Leighty and M.T. Takahashi. 1972. Evaluation of herbicides for possible mutagenic properties. J. Agric. Food Chem. 20: 649-656.

Aulicino, F., M. Bignami, A. Carere, G. Conti, G. Morpurgo and A. Velcich. 1976. Mutational studies with some pesticides in Aspergillus nidulans. Mutat. Res. 38: 138.

BCPC (British Crop Protection Council). 1977. Pesticide Manual. BCPC, Worcestershire, England. p. 334.

Bogyo, D., D.A. Gray, K. Foote, A. Rosenberg, R. Sugatt and D. Orzel. 1981. Criteria Document for 2,4-Dichlorophenoxyacetic Acid. Environmental Criteria and Assessment Office, Cincinnati, OH.

Bollag, J.M., C.S. Helling and M. Alexander. 1967. Metabolism of 4-chloro-2-methylphenoxyacetic acid by soil bacteria. Appl. Microbiol. 15(6): 1393-1398.

Carere, A., G. Cordamone, V. Ortali, M.L. Bruzzone and G. DiGiuseppe. 1976. Mutational studies with some pesticides in Streptomyces coelicolor and Salmonella typhimurium. Mutat. Res. 38: 136.

Cornet, M. 1968. New adventive weed in French rice fields, Potamogeton natans. C.R. Journees Etud. Herbic., Conf. 1: 239-247. (Fre.)

Faulkner, J.K. and D. Woodcock. 1965. Fungal Detoxication. Part VII. Metabolism of 2,4-dichlorophenoxyacetic and 4-chloro-2-methylphenoxyacetic acids by Aspergillus niger. J. Chem. Soc. 1187-1191. MRID. 005003766. (Cited in U.S. EPA, 1982)

Fisher, P.R., J. Appleton and J.M. Pemberton. 1978. Isolation and characterization of the pesticide-degrading plasmid pJP1 from Alcaligenes paradoxus. J. Bacter. 135(3): 798-804. (Cited in U.S. EPA, 1982)

Flint, G.W., J.J. Alexander and O.P. Funderburk. 1968. Vapor pressures of low-volatile esters of 2,4-D. Weed Sci. 16: 541-544. (Cited in NRCC, 1978)

Frank, R., G.J. Sirons and B. Ripley. 1979. Herbicide contamination and decontamination of well waters in Ontario, Canada, 1969-78. Pestic. Monit. J. 13(3): 120-127.

Gamar, V. and J.K. Gaunt. 1971. Bacterial metabolism of 4-chloro-2-methylphenoxyacetate. Formation of glyoxylate by side-chain cleavage. Biochem. J. 122(4): 527-531.

Gaunt, J.K. and W.C. Evans. 1971a. Metabolism of 4-chloro-2-methylphenoxyacetate by a soil pseudomonad. Ring-fission, lactonizing, and delactonizing enzymes. 122(4): 533-542.

Gaunt, J.K. and W.C. Evans. 1971b. Metabolism of 4-chloro-2-methylphenoxyacetate by a soil pseudomonad. Preliminary evidence for the metabolic pathway. Biochem. J. 166(4): 519-526.

Grover, R., L.A. Kerr, K. Wallace, K. Yoshida and J. Maybank. 1976. Residues of 2,4-D in air samples from Saskatchewan: 1966-1976. J. Environ. Sci. Health. B11(4): 331-347. (Cited in Bogyo et al., 1981)

Gurd, M.R., G.L.M. Harmer and B. Lessel. 1965. Summary of toxicological data. Acute toxicity and 7-month feeding studies with mecoprop and MCPA. Food Cosmet. Toxicol. 3: 883-885.

Hamaker, J.W. and H.O. Kerlinger. 1969. Vapor pressure of pesticides. Adv. Chem. Ser. 86: 39-54. (Cited in NRCC, 1978)

Helling, C.S. 1971. Pesticide mobility in soils. II. Applications of soil thin-layer chromatography. Soil Sci. Soc. Am. Proc. 35: 737-743.

Helling, C.S. and B.C. Turner. 1968. Pesticide mobility: Determination by soil thin-layer chromatography. Science. 162(3853): 562-563.

Hendry, D.G. and R.A. Kenley. 1979. Atmospheric reaction products of organic compounds. U.S. EPA, Washington, DC. EPA 560/12-79-001.

Herzel, F. and G. Schmidt. 1979. Testing the leaching behavior of herbicides on lysimeters and small columns. WaBoLu-Berichte. (3): 1-16. (Cited in U.S. EPA, 1982)

Jensen, D.J. and E.D. Schall. 1966. Determination of vapor pressures of some phenoxyacetic herbicides by gas-liquid chromatography. J. Agric. Food Chem. 15: 123-126. (Cited in NRCC, 1978)

Jensen, H.L. and H.I. Petersen. 1952a. Detoxication of hormone herbicides by soil bacteria. Nature (London). 170(4314): 39-40. (Cited in U.S. EPA, 1982)

Jensen, H.L. and H.I. Petersen. 1952b. Decomposition of hormone herbicides by bacteria. Acta Agriculturae Scandinavica. 2: 215-231. (Cited in U.S. EPA, 1982)

Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. Ecotoxicol. Environ. Safety. 4: 26-38.

Kilpi, S., V. Backstrom and M. Korhola. 1980. Degradation of 2-methyl-4-chlorophenoxy acetic acid (MCPA), 2,4-dichlorophenoxyacetic acid (2,4-D), benzoic acid and salicylic acid by Pseudomonas sp. HV3. FEMS Microbiol. Lett. 8(3): 177-182.

Kilpi, S., V. Backstrom and M. Korhola. 1983. Degradation of catechol, methylcatechols and chlorocatechols by Pseudomonas sp. HV3. FEMS Microbiol. 18(1-2): 1-5.

Kirkland, K. and J.D. Fryer. 1972. Degradation of several herbicides in a soil previously treated with MCPA. Weed Res. 12(1): 90-95.

Kybett, B.D., J. Chase and R. Grover. 1976. Vapor pressures of some common 2,4-D esters. (Unpublished data.) (Cited in NRCC, 1978)

Linden, E., B.E. Bengtsson, O. Svanberg and G. Sundstrom. 1979. The acute toxicity of 78 chemicals and pesticide formulations against two brackish water organisms, the bleak (Alburnus alburnus) and the harpacticoid Nitocra spinipes. Chemosphere. 8(11): 843-851.

Loos, M.A., I.F. Schlosser and W.R. Mapham. 1979. Phenoxy herbicide degradation in soils: Quantitative studies of 2,4-D and MCPA-degrading microbial populations. Soil Biol. Biochem. 11(4): 377-385.

Mill, T. 1980. Hydrolysis and oxidation processes in the environment. Soc. Environ. Toxicol. Chem., 1st Ann. Meet., November 23-25. SETAC paper. (Cited in Bogyo et al., 1981)

Moriya, M., T. Ohta, K. Watanabe, T. Miyazawa, K. Kato and Y. Shirasu. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116(3-4): 185-216.

Nakajima, S., N. Naito and T. Tani. 1973. Microbial transformation of 2,4-D and its analogues. Chem. Pharmacol. Bull. 21(3): 671-673. MRID-005005548. (Cited in U.S. EPA, 1982)

NIOSH (National Institute of Occupational Safety and Health). 1983. RTECS (Registry of Toxic Effects of Chemical Substances). November: online.

NRCC (National Research Council Canada). 1978. Phenoxy herbicides -- their effects on environmental quality with accompanying scientific criteria for 2,3,7,8-tetrachlorodibenzodioxin (TCDD). Environmental Secretariat, Ottawa, Canada. Publ. No. NRCC 16075.

NTP (National Toxicology Program). 1983. Management status report dated December 5.

Parry, J.M. 1973. The induction of gene conversion in yeast by herbicide preparations. Mutat. Res. 21: 83-91.

Prescott, L.F., J. Park and L. Darrien. 1979. Treatment of severe 2,4-D and mecoprop intoxication with alkaline diuresis. Br. J. Clin. Pharmacol. 7(2): 111-116.

Que Hee, S. and R.G. Sutherland. 1979. Vapor and liquid phase photolysis of the n-butyl ester of 2,4-D. Arch. Environ. Contam. Toxicol. 8: 247-254.

Reisinger, L. and E. Robinson. 1976. Long-distance transport of 2,4-D. J. Appl. Meteor. 15: 836-845. (Cited in Bogyo et al., 1981)

Reisinger, L. and E. Robinson. 1977. Reply: To comments from (Decker-Hennessey, 1977) on paper by (Reisinger and Robinson, 1976). J. Appl. Meteor. 16: 1009-1010. (Cited in Bogyo et al., 1981)

Rodgers, C.A. and D.L. Stalling. 1972. Dynamics of an ester of 2,4-D in organs of three fish species. Weed Sci. 72(20): 101-105.

Sattar, M.A. 1981. Persistence of 4-chloro-o-cresol and 5-chloro-3-methyl-catechol in soil. Chemosphere. 10(9): 1011-1017.

Sattar, M.A. 1982. Persistence of 4-chloro-2-methylphenoxyacetic acid in soil. Bull. Environ. Contam. Toxicol. 28(3): 348-352.

Sattar, M.A. and J. Paasivirta. 1980. Fate of chlorophenoxyacetic acids in acid soil. Chemosphere. 9(12): 745-752.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1. Toxicology Data. U.S. EPA, Washington, DC. EPA 540/9-79-003A. NTIS PB 80-176876.

Seiler, J.P. 1979. Phenoxyacids as inhibitors of testicular DNA synthesis in male mice. Bull. Environ. Contam. Toxicol. 21(1-2): 89-92.

Smith, A.E. 1982. Soil persistence studies with [14C] MCPA in combination with other herbicides and pesticides. Weed Res. 22(3): 137-142.

Smith, A.E. and B.J. Hayden. 1980. Hydrolysis of MCPA esters and the persistence of MCPA in Saskatchewan soils. Bull. Environ. Contam. Toxicol. 25(3): 369-373.

Smith, A.E. and B.J. Hayden. 1981. Relative persistence of MCPA, MCPB and mecoprop in Saskatchewan soils, and the identification of MCPA in MCPB-treated soils. Weed Res. 21(3-4): 179-183.

Soulas, G., P. Codaccioni and J.C. Fournier. 1983. Effect of cross-treatment of the subsequent breakdown of 2,4-D, MCPA and 2,4,5-T in the soil. Behavior of the degrading microbial populations. Chemosphere. 12(7-8): 1101-1106.

Soderquist, C.J. and D.G. Crosby. 1975. Dissipation of 4-chloro-2-methylphenoxyacetic acid (MCPA) in a rice field. Pest. Sci. 6(1): 17-33. (Cited in U.S. EPA, 1982)

SRI (Stanford Research Institute). 1976. Chemical Economics Handbook. Herbicides. SRI, Menlo Park, CA. p. 573.7008G.

Stark, J. 1983. Persistence of herbicides in forest soils. Weeds Weed Control. 24(1): 275-286.

Steenson, T.I. and N. Walker. 1956. Observations on the bacterial oxidation of chlorophenoxy acetic acids. Plant Soil. 8: 17-32.

Stevens, T. and R.B. Grorud. 1979. High pressure liquid chromatography of ester and salt formulations of 2-methyl-4-chlorophenoxy acetic acid. J. Assoc. Off. Anal. Chem. 62: 738-741.

Strekozov, B.P., G.K. Gryzlova, A.L. Chimishkyan, et al. 1979. Transformations of herbicides in water, air, and soil. Khim. Sel'sk. Khoz. 17(12): 28-31.

Torstensson, N.T., J. Stark and B. Goransson. 1975. Effect of repeated applications of 2,4-D and MCPA on their breakdown in soil. Weed Res. 15(3): 159-164.

U.S. EPA. 1982. MCPA (4-chloro-2-methylphenoxyacetic acid) Pesticide Registration Standard. Office of Pesticides and Toxic Substances, Washington, DC.

Verschuuren, H.G., R. Kroes and E.M. Den Tonkelaar. 1975. Short-term oral and dermal toxicity of MCPA and MCPP. Toxicology. 3(3): 349-359.

Virtanen, M., M.L. Hattula and A.U. Arstila. 1979. Behavior and fate of 4-chloro-2-methylphenoxyacetic acid (MCPA) and 4,6-dichloro-o-cresol as studied in an aquatic-terrestrial model ecosystem. Chemosphere. 8(7): 431-442.

Zepp, R., N.L. Wolfe, J.A. Gordon and G.L. Baughman. 1975. Dynamics of 2,4-D esters in surface water: Hydrolysis, photolysis and vaporization. Environ. Sci. Technol. 9(13): 1144. (Cited in Bogyo et al., 1981)

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

These searches were conducted in November, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7th), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. 486 p.

ACGIH (American Conference of Governmental Industrial Hygienists). 1983. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-84. Cincinnati, OH. 94 p.

BCPC (British Crop Protection Council). 1977. Pesticide Manual, 5th ed., H. Martin and C.R. Worthing, Ed. British Crop Protection Council. 593 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. 2879-3816 p.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. 3817-5112 p.

Grayson, M. and D. Eckroth, Ed. 1978-83. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Vol.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., MA. 575 p.

ITII (International Technical Information Institute). 1982. Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal with Toxicity and Hazard Data. ITII, Tokyo, Japan. 700 p.

NTP (National Toxicology Program). 1983. Carcinogenesis Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, I.N. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nos Reinhold Co., NY.

SRI (Stanford Research Institute). 1983. Directory of Chemical Producers. Stanford, CA.

U.S. EPA. 1982. Chemical Activities Status Report, 3rd ed. (EPACASR). Offices of Pesticides and Toxic Substances, Washington, DC. EPA 560/TIIS-82-002b.

U.S. EPA. 1983. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call In Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1983. CHIB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (United States International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982. Washington, DC. USITC Publ. 1422.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.